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Letters to the Editor

Dear Editor

Tuberculous empyema presenting after oleothorax

Forouhi and colleagues (1) describes a 78-year-old man who presented in 1989 with a discharging chest wall sinus and an associated pleural effusion. He had originally been treated in 1927 with an oleothorax after failure of pneumothorax therapy for pulmonary tuberculosis. They aspirated his pleural fluid, which was mineral oil; on culture it yielded *Mycobacterium tuberculosis*, sensitive to the usual chemotherapeutic agents. Despite treatment, he died.

The authors attribute his death to recognized complications of oleothorax therapy, but might not the entire illness have been avoided by a year of prophylactic isoniazid or other suitable chemotherapy, administered sometime in the intervening decades? Isn't the real cause of death a failure of public health practice?

Are there other untreated 'old tuberculars', similarly at risk?

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Dear Editor

Analgesia and sedation in fibre optic bronchoscopy

We read with interest the recent short report by Williams *et al.* on the acceptability of intravenous midazolam for fibre optic bronchoscopy (1). However we are somewhat surprised by the method in which they undertake the procedure. It is well recognized that the topical nasal anaesthesia is perceived as the most unpleasant part of the procedure (2), although this may be improved by using lignocaine gel (3–5).

An alternative approach which we routinely follow is to give the intravenous sedation prior to application of 4% lignocaine solution via a Mackintosh atomizer. The nares are sprayed and after a few minutes to allow for anaesthesia, the atomizer is placed in the nostrils and the patient is encouraged to sniff during spraying, this is usually followed by a small cough. Good nasal anaesthesia is achieved and patients appear to cough less when the cords are formally sprayed via the bronchoscope, probably as the nasal lignocaine aerosol has already partially anaesthetized the larynx. Moreover as the patient is already sedated they have no recollection of either the spraying of the nose or passage of the bronchoscope.

The technique we describe is clean and simple to follow, allows a clear view, produces effective anaesthesia and is exceptionally well tolerated. We would recommend this technique of upper airway anaesthesia and suggest that it is logical to use sedation prior to the most unpleasant part of the procedure.

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Dear Editor

Treatment of asthma in childhood

In their recent paper (1) Agertoft and Pedersen describe 216 children with mild and moderate asthma who were followed for 1–2 yr (run-in) before being given budesonide (BUD) for 3–6 yr. The dose of BUD started at 800 μg day^{-1} and was titrated up or down according to the degree of asthma control. The children receiving BUD were improved compared both with run-in and with a group of 'control'

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children who were selected on the basis of choice of treatment, their parents not wanting their child to receive inhaled corticosteroid 'because they were afraid of side effects.' Since this was not a randomized study, the comparison with the 'control' group could be biased in either direction.

On the basis of results in the run-in and 'control' groups, the authors suggest that 'suboptimally treated asthma may result in irreversible airway obstruction' (though no attempt was made to assess reversibility). Their treatment consisted of theophylline (56 and 64% during run-in and 'control', respectively), sodium cromoglycate (40%, 58%) and oral β_2-agonist (46%, 42%); it is not clear whether these children had access to inhaled β_2-agonist. These treatments were given throughout the study 'according to the normally recommended guidelines' as they existed in 1989 (2), although these have since been superseded by more recent recommendations (3–5). We agree that the treatment in these groups is, by present standards, suboptimal. It would, however, be incorrect to imply from these results that treatment with sodium cromoglycate could lead to an 'irreversible' loss in lung function. König (6) recently described a favourable long term outcome after treating children optimally for 3–14 yr with sodium cromoglycate. König, who did measure reversibility, showed improved or normal pulmonary function and 'no evidence of irreversible obstruction after up to 14 yr of follow-up.' These results support earlier findings (7).

Agertoft and Pedersen's study cannot be regarded as a direct comparison of BUD and sodium cromoglycate, nor does it show that lung function, or any other aspect of asthma, deteriorates with sodium cromoglycate. Further, it does not assess the effectiveness of *current* treatment recommendations for children with asthma. All Agertoft and Pedersen did show were the benefits of treating poorly controlled asthma with intensive anti-inflammatory therapy.

The authors state that, after starting on BUD, no children required sodium cromoglycate and the use of theophylline and oral β_2-agonists was significantly reduced. This is not surprising since, according to the study design, treatment with sodium cromoglycate was always stopped when BUD was started. Theophylline and/or oral β_2-agonists were only added if high-dose BUD did not result in good control of asthma, and in such children the BUD dose could be increased. The number of children requiring an increase in dose is not stated although a high-dose (>800μg; mean 1025 $\mu g\,day^{-1}$) group was analysed for the effect of BUD on growth.

On growth, analysis of the annual change in height standard deviation scores (SDS) showed that, compared with run-in and low doses ($\leq 400\,\mu g\,day^{-1}$), 'high' doses of BUD ($>400\,\mu g\,day^{-1}$) were associated with a significant reduction in the change in height SDS. As with symptoms and lung function, the comparison of effects on growth is not between BUD and sodium cromoglycate treatment but between well and poorly controlled (undertreated) asthma. It is well known that asthma, if not well controlled, will itself stunt growth. The SDS analysis does not include data from normal children, although a separate group of normal children was analysed for other growth variables. The final, 4-yr mean daily doses were 447 μg from Turbuhaler and 612 μg from Nebuhaler; thus, both devices are delivering doses above the safe cut-off point, as defined in the paper, of 'up to $400\,\mu g\,day^{-1}$'.

Finally, the authors wonder whether BUD will help the children to grow out of their asthma. The clinical benefits described in these children probably depend upon continued intake of inhaled steroid. A recent study (8,9) showed that although 60% of children had a remission of symptoms during up to 3 yr of treatment with BUD $600\,\mu g\,day^{-1}$, two-thirds later relapsed and at the end of follow-up only about half of these had normal lung function, while airway hyperresponsiveness remained abnormal in the majority of patients. When BUD was tapered off over 2 months and discontinued in some children, their asthma deteriorated rapidly. At the end of 6 months, airway calibre and responsiveness had fallen to the same levels as before the 3 yr of BUD treatment; children who were in a complete remission appeared to decline as rapidly as those who had not normalized on inhaled steroid.

Asthma is probably a lifelong problem and the risks of long-term treatments must be weighed against their benefits. In this context Agertoft and Pedersen's study can only be regarded as short term and highlights the need for well controlled, long term studies of intervention in childhood asthma (10).

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Dear Editor

Fulminant psittacosis

The genus Chlamydiaceae consists of three species, all of which may cause disease in humans; *Chlamydia pneumonia, C. psittaci* and *C. trachomata. C. pneumonia*, first isolated in the 1960s, described as the TWAR agent in 1986 and considered a strain of *C. psittaci* at that time, has since been recognized as a separate species, and therefore may account for a significant proportion of previously-diagnosed cases of *C. psittaci* infection (1).

We wonder whether the recent case report of acute renal failure due to *C. psittaci* (2) was in fact due to *C. pneumonia*. We had experience of a similar case of a 61-year-old man who had been exposed to cages containing bird excrement and plumage, while repairing pipes in a pet shop. Our patient developed acute renal failure associated with a respiratory infection. After a period of assisted ventilation, renal replacement with dialysis and antibiotic therapy, he made a complete recovery with normalization of his renal function. Antibody titres, using complement fixation, to Chlamydia group antigen confirmed a significant rise from 1/10 to 1/80, but species-specific serology of the sample indicated *C. pneumonia* with a titre of 1/10280 and a titre of 1/280 for *C. psittaci*, due to cross reactivity with common antigen. Therefore, despite the strong epidemiological history, a diagnosis of psittacosis was excluded.

It is clear that the diagnosis in this case, and hence the reason for reporting the case, hangs on the confirmation of psittacosis. The authors give no titres regarding species-specific serology of Chlamydia group antigen, and we wonder whether it would be helpful to ascertain the exact cause of the initial infection using species-specific serology, recognizing its limited availability at present.

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